

University of Groningen

TNF signaling in non-alcoholic fatty liver disease

Aparicio-Vergara, Marcela

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version

Publisher's PDF, also known as Version of record

Publication date:

2013

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Aparicio-Vergara, M. (2013). *TNF signaling in non-alcoholic fatty liver disease*. [Thesis fully internal (DIV), University of Groningen]. [S.n.].

Copyright

Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: <https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment>.

Take-down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): <http://www.rug.nl/research/portal>. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

Centrale	U
Medische	M
Bibliotheek	C
Groningen	G

Stellingen

Behorende bij het proefschrift

TNF Signaling in Non-Alcoholic Fatty Liver Disease

Marcela Aparicio Vergara

September 18th 2013

1. The discrepancy between studies using a "loss-of-function" (Uysal *et al.* 1997, Schreyer *et al.* 1998, Pamir *et al.* 2009, Toda *et al.* 2010) and a "gain-of-function" mutation in the TNF receptor (this thesis) highlights our simplistic understanding of the role of TNF signaling in insulin resistance.
2. Given that ALT/AST levels are persistently normal in over half of the patients with NAFLD (Fracanzani *et al.* 2008) and in rodent models of NASH (this thesis), novel biomarkers are needed to identify NAFLD.
3. The reported dissociation between the development of macrophage-associated inflammation and the onset of insulin resistance (Turner *et al.* 2013, this thesis) questions the causality of the alleged association between inflammation and insulin resistance.
4. Given that all the immune cells have been replaced in a bone marrow transplantation experiment the interpretation of many bone marrow transplantation studies can be complicated. (Duran-Struuck *et al.* 2009, this thesis)
5. Despite all our efforts to stay lean, the obesity paradox implies that we need the extra pounds in later life. (Hughes 2013)
6. Although they have a reputation of being the healthiest and most active generation, baby boomers are actually in worse overall health than their parents. (King *et al.* 2013)
7. The same factor (TNF) in our immune system that is instrumental in enabling us to fight off severe and dangerous inflammatory ailments is also a player in doing the opposite at a later stage, causing the suppression of our adaptive immune response. (Sade-Feldman *et al.* 2013)
8. A scientist in his laboratory is not a mere technician: he is also a child confronting natural phenomena that impress him as though they were fairy tales. (Marie Curie)
9. Nowadays we have countless ways of communicating, but nothing more to say.
10. Ser científico es una profesión, es un estilo de vida.